Chapter 18

Chemical, Environmental, and Health Aspects of the Seveso, Italy, Accident

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1. INTRODUCTION

Today *dioxin* is a familiar word, but very few people in Italy had heard of it before the Seveso accident of July 10, 1976. Ever since, TCDD and related chemicals[1,2] have been dealt with profusely by the domestic and international scientific communities. Because of the sensitizing effect it had, the Seveso case should be considered a turning point in environmental risk management. For instance, it is recalled here that, in February of 1988, the Commissione Consultiva Tossicologica Nazionale (CCTN)—Italy's National Toxicology Commission—established a set of maximum tolerable limits (MTLs) for polychlorinated dibenzodioxins (PCDDs) and dibenzofurans (PCDFs) in several environmental matrices.[3,4] These limits were based on the 1987 array of U.S. EPA toxicity equivalence factors (TEFs) to convert PCDD and PCDF analytical data into "toxicity equivalents of TCDD" (TE units),[1] and also on much of the technical experience gained by dealing with the Seveso event.

2. THE ACCIDENT OF JULY 10, 1976

2,4,5-Trichlorophenol (TCP) production at the Givaudan–Hoffmann–LaRoche ICMESA plant at Meda (Milan, Italy) was started in 1969–1970 and brought up to full-

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scale levels in the years that followed with a big production increase ($>10^5$ kg/year) over the 1974–June 1976 period. TCP was obtained by a discontinuous process based on hydrolyzing 1,2,4,5-tetrachlorobenzene to sodium trichlorophenate with sodium hydroxide in the presence of xylene and ethylene glycol, and transforming the trichlorophenate to TCP by acidifying it with hydrochloric acid. The hydrolysis reaction was carried out inside ICMESA's Department B, by utilizing a 10-m^3 stainless steel reactor, in 7000-kg batches of chemicals.[5–8]

At 12:37 p.m. on July 10, 1976, an exothermic reaction raised the temperature and pressure inside the reactor beyond limits, thereby causing a safety device, consisting of a rupture disk set at ~3.8 atm (380 kPa), to blow out.[5–9] TCDD production was also increased to an unknown extent. The safety device was mounted on an exhaust pipe that was directly connected to the reactor cover and, passing through the roof of Department B, ended up in the open. When the rupture disk collapsed, the overheated fluid mixture burst through the pipe out into the open air propelled by the thrust of the built-up pressure. The chemical cloud that left the reactor entrained nearly 2900 kg of organic matter, including at least 600 kg of sodium trichlorophenate and an amount of TCDD which is still being evaluated. The visible part of the cloud rose up to about 50 m; it subsequently subsided and fell back to earth, but was wind-driven over a wide area of territory (Fig. 1).[5,10–13]

Emission gradually decreased until it ceased altogether. Within less than 2 hr of the accident, chemicals settled on the ground as far as 6 km south of ICMESA, or were dispersed by wind streams.[6,13,14] Serious environmental contamination followed: the leaves of plants near ICMESA, courtyard animals, and birds were seriously affected, many dying within a few days of the accident. At the same time, dermal lesions among humans who had been exposed to the toxic alkaline cloud began to appear. About 10 days after the accident, TCDD was found in various types of samples collected near the plant.

Toward the end of the runaway reaction, reactor temperatures are thought to have increased well above 300°C, thus causing extensive mineralization of residual organic substances.[5,7,15,16] After blowout, ~2300 kg of residual chemicals was present in the reactor; their approximate composition was sodium chloride (72%), decomposed organic matter, and some 250–300 g TCDD.[7,8,17] TCDD amount was later reestimated at approximately 600 g.

3. RISK MANAGEMENT MEASURES AND ASSESSMENT

3.1. Definition of Areas at Different Risk Levels

As a first step, the information available on the location of toxic and pathological events—regarding vegetation, animals, and humans[18]—and on the airstream pattern at blowout time was used to draw an approximate diagram of the contaminated area. This was further confirmed by chemical monitoring of TCDD in the soil carried out under emergency conditions.[6,13,19–21] Within 5 weeks of the accident, the area hit was

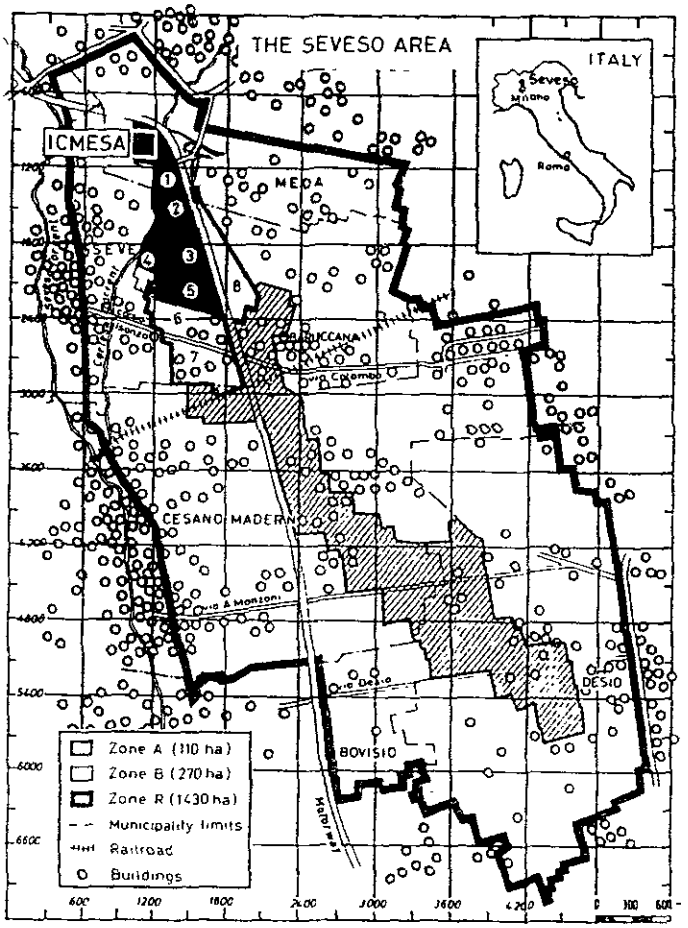

Figure 1. The contaminated area at its maximum extension. Zone A fanned out from ICMESA to the south and contained most of the TCDD that had escaped. Zone B was the natural extension of Zone A along the main diffusion pathway of the TCDD-containing cloud. Both Zones A and B were enclosed by a larger territory coded as Zone R. The grid unit cell was a 50-m square for Zone A and a 150-m square for Zones B and R. Numerals identify Subzones A_1–A_8. (Adapted from di Domenico *et al.*[13])

subdivided into Zones A, B, and R, in descending TCDD and toxicological risk levels (Fig. 1).

The borderline between Zone A and Zone B was set at average TCDD concentrations in the soil of $\leq 50\ \mu g/m^2$; the boundaries of Zones A and B in Zone R were fixed where average contamination was $\leq 5\ \mu g/m^2$. Zone R included the remaining territory where detectable levels of TCDD (formally, $\geq 0.75\ \mu g/m^2$) were found. In all cases, borderlines were established following preexisting natural or artificial divisions, in general compliance with the contamination pattern made out.[5,6,8,13,19,22] Since TCDD concentration detected in Zone A ranged over more than four orders of magnitude

Table 1
Summary of Some Analytical Features of Zones A, B, and R, and Breakdown of Zone A into Subzones A_1–A_8[a]

	TCDD levels (μg/m²) according to different mappings[b]				
		January 1977[c]			
Zone A subzone	September 1976 (means)	Means	Ranges	March 1978[c] (means)	1979–1980 (means)
A_1	1.6 E+3[d]	4.5 E+2	<0.75–5.5 E+3	6.2 E+2	9.9 E+2
A_2	2.5 E+3	4.3 E+2	6.1–1.7 E+3	4.0 E+2	1.2 E+3
A_3	1.3 E+2	3.5 E+2	<0.75–2.0 E+3	1.6 E+2	1.9 E+2
A_4	2.6 E+2	1.7 E+2	<0.75–9.0 E+2		1.1 E+2
A_5	1.2 E+2	6.3 E+1	<0.75–4.3 E+2		7.4 E+1
A_6	9.1 E+1	3.1 E+1	<0.75–2.7 E+2		
A_7	1.2 E+1	1.7 E+1	<0.75–9.2 E+1		
A_8[e]	<5.0				
Zone B	1976–1977 (mean)		1978 (mean)	1980–1981 (mean)	1983 (mean)
	3.4		4.3	3.2	2.8
Zone R	~0.5[f]				

[a] From di Domenico *et al.*,[43] La Porta *et al.*,[30] and Reggiani.[8]
[b] The configuration of Subzones A_1–A_7 changed with time and the data reported provide only broad indications of area-specific contamination levels. Mappings identified according to di Domenico *et al.*[43] and La Porta *et al.*[30]
[c] These mappings are now considered to be biased. Yet they are very important as examples of early Zone A investigations based on a 50-m-square grid and because of the many soil site checks carried out.
[d] 1.6 E+3 = 1600
[e] Because of the low TCDD concentrations, Subzone A_8 was soon reclassified as part of Zone B.
[f] Fading into background with time elapsing and agronomical works.

(from <0.75 μg/m² to >20 mg/m²),[8,13,23] the zone was broken down into Subzones A_1–A_8, each characterized by a somewhat lower range of TCDD levels (Table 1).

On July 26, 1976, approximately 200 people were evacuated from a 15-ha area immediately southeast of ICMESA[5,6,8,13,22,24–26]—the first part of what shortly afterward would be defined as Zone A. Following further analytical findings concerning the TCDD contents of soil and vegetation samples, a few days later the entire Zone A (over 730 inhabitants altogether) was evacuated. Zones B and R were subjected to area-specific hygiene regulations including prohibition of farming, consuming local agricultural products, and keeping poultry and other animals (Table 2). Indeed, many major risk management measures and policies were already established during the emergency period (July 10–August 15, 1976).

3.2. TCDD Contamination Maps

Beginning with the emergency period, soil monitoring was performed repeatedly during the first decade following the Seveso incident (1976–1986) for at least three

Table 2
Measures Designed to Limit Human Exposure in the Seveso Area[a]

Measures affecting people's behavior
Evacuation of Zone A
To avoid any contact with highly contaminated soil, some 160 houses in Zone A were evacuated
Hunting prohibited for approximately 8 years
Precautions for Zone B residents
Intensification of personal hygiene
No animal breeding or vegetable planting
Daily relocation of children up to 12 years old and pregnant women
Abstention from procreation
Minimization of air dust level (vehicle speed limit, 30 km/hr)
Careful emptying of vacuum cleaners
Hunting prohibited for approximately 8 years
Precautions for Zone R residents
Intensification of personal hygiene
No animal breeding or vegetable planting
Pets to be fed with food from areas other than Zones A, B, and R
Hunting prohibited for approximately 8 years
Measures acting on the environment
Measures common to all zones
All locally bred animals (mostly chickens and rabbits) slaughtered and their carcasses transferred to Zone A
Honey collected and disposed of in Zone A
Defoliation and agronomic activities
Defoliation aimed at removing TCDD deposited on grass and leaves; before cutting, green parts sprayed with vinyl acetate glue to fix TCDD
Ploughing performed repeatedly to dilute TCDD in topsoil layer and facilitate its dissipation
Reclamation of Subzones A_6 and A_7
Cleanup carried out by four teams, each working on a 4-hr/day shift; workers wore individual protective equipment
Schools
Thorough interior cleanup and floor washing daily
Monthly analytical checking on indoor contamination status for over a year after the accident. Procedure discontinued after TCDD levels consistently found <0.01 $\mu g/m^2$ (tolerable limit)

[a] Adapted from Fortunati and La Porta [26]

reasons: for reassessment of TCDD distribution patterns and levels with time, reassessment and updating of risk estimates and risk management measures, and as a backup tool to determine the effectiveness of remedial actions and reclaiming operations. Thousands of soil samples were collected and assayed according to criteria, techniques, and procedures often developed ad hoc to meet the requirements of a unique and unexpected case for which no reference to a former experience in Italy could be made. In general, the analytical tools and setups grew more sophisticated and reliable as time went by[13,14,23,24,27]; however, studies have proven that the sets of soil data up to 1979 may have seriously underestimated TCDD levels.[23,28,29]

Aside from the Zone A map of September, 1976, for which sampling was carried

out on a polar coordinate reference system, all other mappings were obtained by establishing sampling sites on a north–south grid.[5,13,20,30,31] Generally, sampling frequency provided one datum per cell, although cases of higher frequency are often encountered. The usual way of building and representing a contamination map was to asssociate the magnitude of the analytical outcomes to graphic symbols (for instance, a larger dot for a higher TCDD level) classified by discrete ranges, and thereby make a graphic report by using the pertinent reference system. However, data sets were also constructed to exhibit TCDD levels as bidimensional isoconcentration curves or histograms (Figs. 2 and 3),[19,32–35] or tridimensional histograms or curves.[28,31,34,36] In

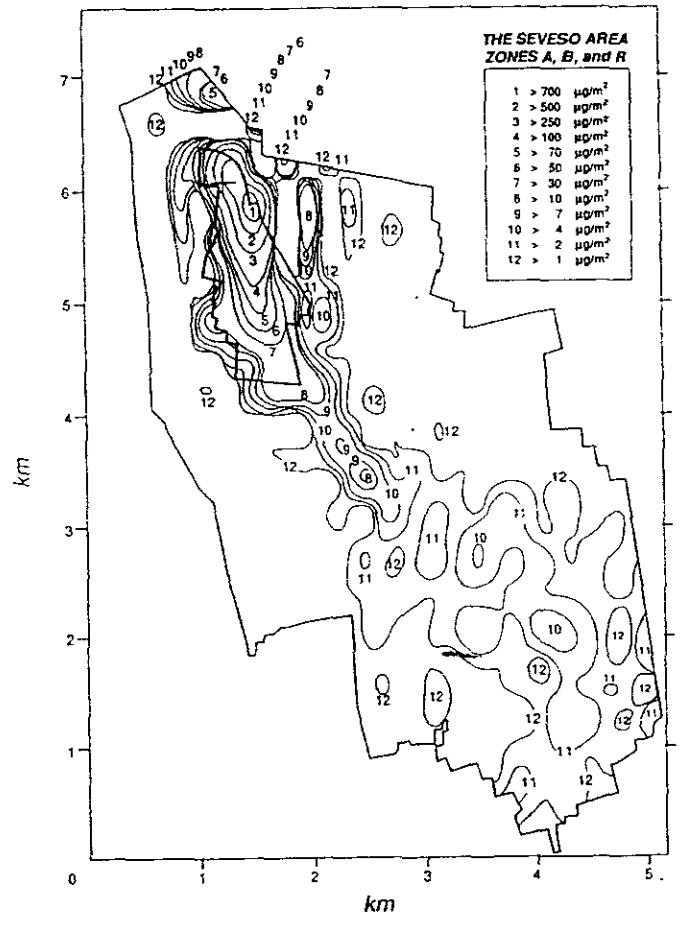

Figure 2. TCDD isoconcentration curves drawn on the basis of approximately 1100 soil measurements carried out in 1976–1977, large areas void of curves are associated with lack of data. Zone A profile is visible within the larger Zone R boundaries. The picture visualizes to some extent the ups and downs of TCDD distribution, greater in Zone A than elsewhere. (Adapted from Belli *et al.*[32])

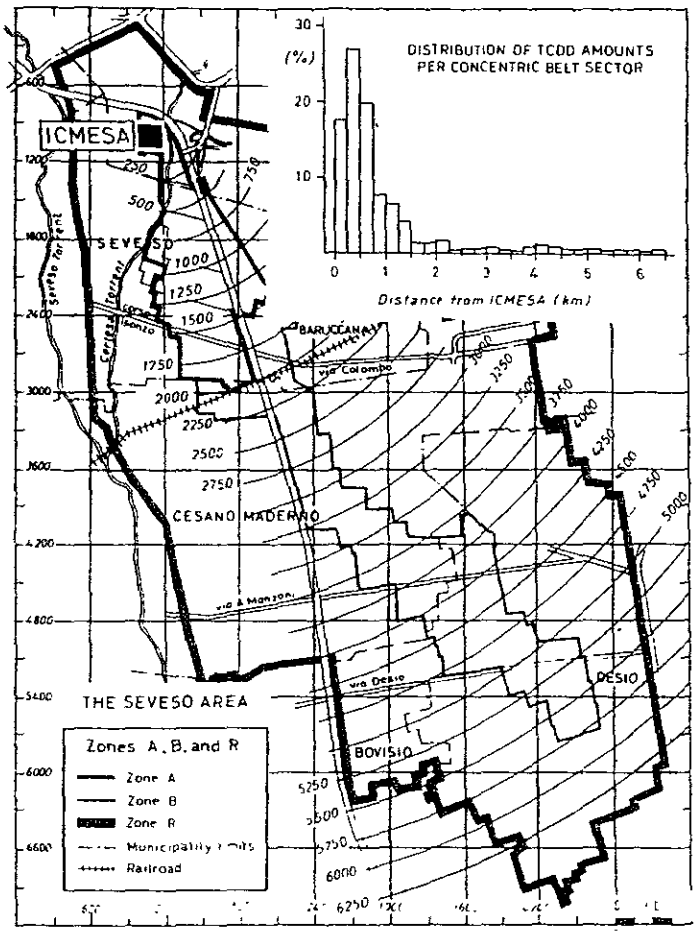

Figure 3. The contaminated area was sliced all the way into 250-m concentric belt sectors centered on ICMESA. TCDD measurements were summed up to yield sector-specific total amounts whose normalized (100%) sum provided the distribution graph in the upper right corner. The histogram shows the exponential-like decrease of TCDD levels in soil with increasing distance from source.

particular, Belli *et al.*[37,38] have recently provided a preliminary tridimensional representation by means of fractal models, currently under development.

As mentioned, contamination maps served different purposes. By integrating concentration levels in soil, 0.1- to 3-kg estimates of total TCDD present within Zone R boundaries were obtained[8,13,19,21,33]; however, values < 0.5 kg were prefered. It should be emphasized that, because of the biases on soil data sets as previously stated, all such figures may well be underestimated by one to two orders of magnitude. Lastly, of the total TCDD amount analytically estimated, 40 to 50% was found to be present within 0.5 km from ICMESA, > 85% occurred within Zone A boundaries, and > 99% was recovered within Zone A and Zone B boundaries.

3.3. Early Assessment and Management of Risk

The first risk assessment for the Seveso area was carried out in order to establish a set of tolerable environmental limits assuming that TCDD intake from ingestion of contaminated soil was the most important factor of hazard. At this stage, several toxicological features of TCDD were already known[8]; however, the risk assessment and management were substantially based on the following issues[39]:

- A no-observed-effect level (NOEL) from subchronic and carcinogenicity studies in rats of 1 ng/day per kg bw[40,41]
- A safety factor of 1000
- Of all inhabitants, children most liable to be at risk
- A tolerable daily intake (TDI) estimated at 1 pg/kg bw

Since reclamation works in farmable soil were aimed at restoring average TCDD concentrations to $< 0.75\ \mu g/m^2$, it was estimated that the adopted TDI would allow a 20-kg child a daily ingestion of up to ~3 g (!) of soil. In the absence of sound data, the bioavailability of soilborne TCDD was conservatively[42] set at 100%.

In light of the above, tolerable limits for land (soil), housing interiors, equipment, and other matrices and items were set by regional law. Definition of risk areas—namely, Zones A, B, and R—was carried out by taking into account the TCDD levels detected predominantly in the 7-cm topsoil layer, and therefore extensive use of the "surface density" unit $\mu g/m^2$ was made, *the unit surface being defined as a 1-m square with a 7-cm thickness*. TCDD surface densities were converted to the commoner ng/kg concentration units multiplying by an average factor of 8. The following was obtained for the different risk areas and matrices[5,13,22,39,43].

Farmable land	$<0.75\ \mu g/m^2$	>6 ng/kg
Not farmable land	$\leq 5\ \mu g/m^2$	≤40 ng/kg
Limit for evacuation	$>50\ \mu g/m^2$	>400 ng/kg
Building outdoor surfaces	$\leq 0.75\ \mu g/m^2$	
Building indoor surfaces	$\leq 0.01\ \mu g/m^2$	

In 1988 these limit values were taken as a reference to set the current maximum tolerable limits. The early risk assessment and management described was focused primarily on defining the area(s) to be evacuated and those subject to hygiene and sanitary restrictions.

3.4. Assessment of Exposure and Risk for Residents of Zones B and R

In Zone B—a typical agricultural–industrial setting with a number of houses that had small private gardens—the original contamination levels were between 5 and 50 $\mu g/m^2$. Since this area was next to the evacuated area and along the main line of contamination, risk assessment for the nearly 5000 people of the zone was regarded as a critical issue by health authorities and local communities. This was partly a result of

risk management policies adopted during the emergency period and based on an analytical layout of TCDD in soil which was incomplete or not fully reliable: indeed, several soil monitorings taken up to 1985 confirmed that the TCDD contamination pattern was very uneven[39] (e.g., see Fig. 2). Because of this, setting the Zone A–Zone B borderline must be considered as somewhat arbitrary.

Therefore, in 1984 a new risk assessment was carried out for Zone B inhabitants (Table 3, upper secton). Compared with that of the emergency period, the new assessment made use of better, more reliable, analytical information and, in particular, of recent data on cancer response in rodents.[41,44–47] A TCDD lifetime daily intake of

Table 3
Examples of Risk and Exposure Assessments Carried out in 1984 for Families of Zone B Having Vegetable Gardens and Courtyard Animals[a]

Risk assessment (per million people)[b]

Exposure route	TCDD intake[c]	Extra cancer risk
Soil,[d] ingestion[e]	4.6 E−3[f]	1.6 E−1
dermal contact	1.7 E−3	6.1 E−2
inhalation[g]	6.0 E−4	2.1 E−2
Drinkable water[h]	≤2.9 E−2	≤1.0
Vegetables	5.7	2.0 E+2
Meat and dairy products	2.4	8.6 E+1
Fish consumption[i]	~0	~0
Totals	8.1	2.9 E+2

Exposure assessment (TCDD intake)[c,j]

Exposure group[k]	Vegetables	Courtyard animals	Other animals	Total[l]	Total[m]
Group 1				3.5 E−2	~0.007
Group 2	3.5			3.5	~0.7
Group 3	3.5	6.7		1.0 E+1	2.0
Group 4	3.5	6.7	2.3 E+1	3.3 E+1	6.7

[a] Model and assumptions: mean TCDD level in most Zone B soil, 1.5 μg/m² (12 ng/kg), average individual weight, 70 kg; length of lifetime exposure, 70 years.
[b] From Fortunati.[39]
[c] In pg/day per kg bw.
[d] Intake and risk figure breakdown obtained by the authors on the basis of original references[39,48] and the later work by di Domenico.[4] Zone B inhabitants not consuming local food were expected to have only a TCDD intake as per Group 1 below.
[e] The only risk evaluated in 1976.
[f] 4.6 E−3 = 0.0046.
[g] Dust level in air, ≤0.14 mg/m³ (see text).
[h] TCDD level in drinkable water, ≤1 pg/liter (see text).
[i] In Zone B, no surface waters to collect fish.
[j] From Pocchiari *et al.*[25] TCDD intakes from drinkable water, vapors, and local cow and human milk considered negligible or not evaluated for lack of data.
[k] Group 1, people not consuming local food. Group 2, people consuming local vegetables; Group 3, people consuming local vegetables and meat from small courtyard animals; Group 4, people consuming local vegetables and animal food. For all groups, background intake from contaminated soil and dust as per Group 1.
[l] Dissipation of TCDD from soil not considered.
[m] Dissipation of TCDD considered (over a 70-year exposure).

0.28 pg/kg bw was estimated to determine an extra cancer risk of 1×10^{-5} and a simplified linear relationship was assumed to exist between cancer response and low TCDD exposures. According to the model employed, most (80%) of Zone B was characterized by a mean TCDD level of 1.5 $\mu g/m^2$ (12 ng/kg), whereas the remaining 20% portion had a mean contamination level that was 10 times as high; the following exposure routes were considered[25,39,48]:

- Ingestion of soil
- Absorption from dermal contact with soil
- Inhalation of contaminated dust
- Contribution of drinkable water
- Ingestion of vegetables grown in home gardens
- Consumption of animal products (primarily chickens and rabbits) from the area

As the resident population was under 5000 people who were only partly exposed to ingesting contaminated food, the conclusion was reached that the conservatively estimated extra cancer incidence of $\sim 3 \times 10^{-4}$ (Table 3, upper section) could not be observed or that it would not be statistically significant. However, owing to the relatively high value of the risk estimate, a more detailed analysis of various parameters (e.g., the rate of TCDD transmigration from soil to vegetation and vegetables, residents' eating habits, including egg and milk consumption not considered in former estimates) was deemed to be advisable, especially where lack of experimental data had required large extrapolations. Part of the analysis required experimental work such as the use of controlled cultivated plots and animal farms in Zone B.[39] In view of the incremental risk magnitude, Lombardy's Regional Government also made arrangements for operations to dilute and possibly replace the contaminated topsoil layer in vegetable gardens and agricultural areas in order to further reduce the risk from consuming vegetables and farm animals from contaminated areas. At the same time, the agricultural procedures, such as plowing, were expected to facilitate TCDD dissipation from topsoil.

In 1984 a specific study was carried out to estimate Zone B population exposure to TCDD, should hygienic restrictions be removed[25]; the study provided a general backup for the risk assessment described above. The following points were used for assessment:

- Average TCDD level in soil was assumed to be 12 ng/kg.
- The entire population (<5000 people) was broken down into four groups with different exposure potentials.
- Information on local dietary habits and food production was based on an ad hoc survey.
- Food consumption estimates were corrected for fraction of food consumed by people not living in Zone B.
- TCDD levels in different food items estimated from analytical data or from soil–vegetation, soil–animal, and soil–vegetation–animal translocation factors were derived from experimental data.
- TCDD half-life in soil was estimated to be 10 years.

The lower section of Table 3 summarizes the outcomes obtained; figures based on TCDD constant levels in soil appear to be fivefold larger than those taking into account TCDD dissipation. In their work, the authors acknowledged the lack of sound data to describe TCDD distribution in Zone B soil and the relationship between the chemical levels in soil and those in plants and animals, so that a "reasonably conservative" approach in estimating risk had been adopted in order to protect human health. On the whole, the estimates indicated that, as potential TCDD intakes might reach values that could not be disregarded, comprehensive analytical investigation on TCDD levels in vegetable and animal foods from Zone B was considered to be essential before removing the ban on farming and consuming local products. In retrospect, it is interesting to compare the results of Table 3, lower section, with the TDI for TCDD presently adopted in Italy and, for instance, recommended at a consultation of the World Health Organisation (TDI = 10 pg/day per kg bw) which is considerably higher than current U.S. EPA guidelines.[3,4,49]

Assessments of exposure and risk for the inhabitants of Zones B and R were carried out by di Domenico and Zapponi.[50] The study was based on the environmental analytical data available and assumed that TCDD intake through food produced locally was zero because of the existing hygienic restrictions; the exposure period considered was from 6 months after the accident onward. Under such conditions, the extra lifetime cancer risk for the cohort at the highest exposure did not exceed $\sim 1 \times 10^{-5}$.

4. REHABILITATION OPERATIONS

After the emergency period, Lombardy's Regional Government issued a special law and set up the Special Office for Seveso to handle sanitary, social, economic, and organizational problems.[22,39,51] By appointing five task groups acting within the Special Office, five rehabilitative programs were implemented and completed. They covered:

- Contamination control and reclamation operations
- Sanitary control for the population and veterinary assistance
- Social assistance (including schools and replacement of dwellings for the evacuated population)
- Reconstruction of homes and public buildings when not reclaimable
- Economic aid to productive groups and facilities (e.g., artisans, industries) damaged by the incident

The five programs were aimed at normalizing life in the contaminated areas on the basis of benefit-to-cost evaluations taking into account the technical, economical, and social aspects as well as public opinion. As an example, in the following an outline is provided of the procedures adopted to reclaim Subzones A_6 and A_7—characterized by relatively low levels of contamination as compared with other Zone A subzones (Table 1)—and Zones B and R.

Subzones A_6 and A_7 had much social, economical, and political importance in

Table 4
Outline of Cleanup Methods Employed for Rehabilitation of Subzones A_6 and A_7[a]

Building interiors	
Items not reclaimed	Textiles (curtains, carpets, clothing, etc.); furniture with textile upholstery; wooden floors, food products; household appliances; low-valued miscellaneous indoor material. All this material was classified, listed, removed (starting from attics down to living quarters and basements), and transferred as such to Subzone A_5 acting as a temporary storage place.
Items reclaimed	Building interiors were vacuum-cleaned and washed thoroughly with water and detergent (wallpaper was removed). Cleansing liquids were sucked, collected in steel containers, and transferred to Subzone A_5. Walls were repainted and floors, if possible, refinished
	Furniture was vacuum-cleaned and washed with solvent-impregnated cloths (later disposed of in steel containers to be transferred to Subzone A_5) Reclaiming started from higher floors and, in flats, from rooms farthest from entrance. Entrance halls were left until the end. Furniture was refinished
External surfaces	External surfaces were washed with pressurized (60 bar) water. Vegetation was cut, ground, and transferred to Subzone A_5 Topsoil was manually or mechanically removed to a depth of 30 cm or more, scarification was halted when residual TCDD levels at excavation bttom matched the pertinent reference limit. Trucks used in reclaiming were not allowed to run over reclaimed areas and had special routes The soil removed was carefully loaded on trucks to avoid spilling during loading and transfers Operations in reclaimed areas were carried out while keeping dust level in air as low as possible by spraying water.

[a]From La Porta and Fortunati.[51]

that they had accommodated about 67% of the evacuated population.[5,8,22,43,51] Together they covered some 32 ha; their minimum distance from ICMESA was 1200 m. TCDD levels in soil had been found up to 270 $\mu g/m^2$; however, estimated mean values were 31 $\mu g/m^2$ in Subzone A_6 and 17 $\mu g/m^2$ in Subzone A_7 (Table 1). The methodology adopted for reclaiming involved mechanical removal of soil with TCDD from surfaces to reach concentrations within the tolerable limits: for instance, removal of the top 25 cm of soil also removed an average of > 90% of total TCDD present.[52,53] As summarized in Table 4, several cleanup methods were employed.

During reclamation operations, building interiors and exteriors were repeatedly checked by means of wipe and/or scrape testing; topsoil cores were taken in gardens.[13,24,27,43] Approximately 700 assays were carried out on the 87 buildings and surrounding gardens. At the end of reclamation operations, all TCDD levels were below the established tolerable limits.[30,51] The same holds for agricultural and farming areas which were checked by sampling topsoil cores in 56 sites selected on the reference grid. Buildings, gardens, and agricultural and farming areas were finally reconditioned and analytically rechecked at random. When these checks gave favorable outcomes, cleanup operations were considered teminated; at that point, health authorities granted clearance for reentry of evacuated people. The very uneven distribution of TCDD in the soil and on internal and external building surfaces (from <0.01 up to some $\mu g/m^2$) led to the adoption of a specific statistical approach when assessing the effectiveness of reclamation operations.[43]

Rehabilitation of Zones B and R was started in 1977.[5,22] It eventually entailed soil removal in public and private gardens, agricultural treatments of topsoil layers to dilute surface TCDD and help its dissipation, and covering with fresh uncontaminated soil. Indeed, simply by ploughing (and mixing), the TCDD levels in the upper 7-cm soil layer could drop by as much as a factor of 3 to 6.[52,54] The effectiveness of agricultural treatments—e.g., ploughing, harrowing, sowing—was increased by repeating treatments during the same year and over subsequent years.[5,8,22] The rapid effect of dilution was accompanied by the slower dissipation processes which were facilitated by bringing TCDD from deeper to surface topsoil layers. Simple, rather inexpensive, and efficacious ploughing alone was largely employed to rehabilitate areas of agricultural and farming interest.[54]

5. ENVIRONMENTAL IMPACT OF THE ACCIDENT

5.1. TCDD Vertical Distribution in Soil

Many in-field investigations were carried out to study the vertical gradient of TCDD in soil and its permeation capacity under the action of meteoric water.[19,52] In fact, one major concern was that the chemical, by permeating the soil, might endanger the local groundwater system. As indicated, TCDD vertical distribution and mobility also had relevant implications on soil reclamation processes and, in a broad sense, on risk assessment and management. Most investigations were performed in Zone A, as the higher TCDD levels facilitated measurements at greater depths and, by intensifying the eventual permeation process, were expected to make it more liable to be detected. Statistical analysis and mathematical modeling were used by different authors to describe the distribution of TCDD in Seveso soil as a function of depth and time.[29,52,55]

In general, TCDD levels dropped steeply between the depths of 4 to 12 cm, and at a much lesser rate from a depth of ~8 cm downward. Until the fall of 1976, most (>90%) TCDD was generally contained in the first 7–8 cm of topsoil, which is why it became customary to monitor soil by coring an ~7-cm topsoil layer. In 1977, > 75% of TCDD was still on average contained in the top 12-cm layer of soil. At Sites A11 and A12 (Table 5)—as in other cases—the vertical gradient in soil was studied at different times. By comparing the available data in the upper six 4-cm-thick soil layers, it may be seen that distribution patterns obtained from investigations carried out on October 15, 1976, and on April 1, 1977, exhibited significant differences in that some TCDD appeared to have moved from the upper layers downward; no further significant changes were observed in the next 8 months. It may be pointed out that the period of greater downward movement was also characterized by a remarkable amount of local atmospheric precipitation. Out of all of the data available, Sites A11 and A12 of Zone A have been used here as a paradigm for the entire set of data; however, occasional significant deviations were observed from site to site even when close to each other.

Table 5 also summarizes the results of an investigation in five Zone B sites; they appear to match the general distribution pattern described above.[52]

Table 5
Vertical Distribution of TCDD in Soil at Sites A11 and A12 in Zone A Investigated in 1976 and 1977, and at Sites B1–B5 in Zone B Sampled on November 11, 1976[a]

Depth (cm)	TCDD level ($\mu g/m^2$)[b]					
	A11[c]	A11[d]	A11[e]	A12[c]	A12[d]	A12[e]
0–4	1.2 E+3[f]	3 1 E+1	1.6 E+1	1.4 E+3	9.2 E+1	3 4 E+2
4–8	2.0 E+2	3.0	9.1 E−1	1.4 E+2	5.6 E+1	1.5 E+2
8–12	3.3 E+1	1 1	1.2	2.5 E+1	3.7 E+1	5.1 E+1
12–16	2.1 E+1	2 3 E−1	4.7 E−1	<2.0 E−2	4.2 E+1	5.7 E+1
16–20	1.6 E+1	1 4	1.9 E−1	6 4	4.1	4.2 E+1
20–24	1.7 E+1	1.9 E−1	2.5 E−1	3 6	5.7	2.6 E+1
24–28	2.0	2 5	9.0 E−2	<2 0 E−2	3 0	9.7
28–32	<2.0 E−2	8.8 E−1	1.3 E−1	2.5	2.5	7.4
32–36	<2 0 E−2	1.0 E+1	4.0 E−2	6.0 E−1	2.6	1.1
36–40	<2.0 E−2		5.0 E−2	1.1	2 5	7 3 E−1
40–44	<2 0 E−2			<2 0 E−2		

	B1	B2	B3	B4	B5
0–4	5.3	6.2	9.7	6 4	1 2 E+1
4–8	1 0 E−1	<5.0 E−2	5.0 E−1	1.0 E−1	3.0 E−1
8–12	<5.0 E−2	<5.0 E−2	<5 0 E−2	3.0 E−1	4.0 E−1
12–16	<5.0 E−2	<5.0 E−2	2.0	<5.0 E−2	1.0 E−1
16–20	2.0 E−1	<5.0 E−2	<5.0 E−2	<2.0 E−2	<5.0 E−2

[a] From di Domenico *et al.*[52]
[b] Estimated amount of TCDD in a parallelepiped of soil with a 1-m^2 base and height equal to the layer thickness
[c] Sampling on October 15, 1976
[d] Sampling on April 1, 1977
[e] Sampling on December 1, 1977
[f] 1.2 E+3 = 1200

5.2. TCDD Levels in Sediments and in Ground and Surface Waters

From shortly after the accident, sampling and analyses were periodically carried out on surface water streams of the area hit as far south as the River Lambro. This river and the Torrent Seveso are both part of the Milan surface water system. Outcomes were found to be consistently negative for TCDD at a detection threshold <10 pg/liter.[20,24] Monthly determinations conducted on pipeline and ground waters also provided consistent negative results, even when detection sensitivity was ~1 pg/liter.

During the same period, sediment samples from the Torrents Certesa and Seveso were also assayed; the former flowed into the latter after passing through a stretch of the most contaminated sector of Zone A (Fig. 1). Results were positive (on the order of 1 ng/kg) within the first few kilometers downstream from their confluence, but negative in samplings performed farther downstream. The intense rainfalls of the fall of 1976 and the following winter caused Torrent Seveso to repeatedly overflow its em-

bankments at the point of entry into Milan, thus depositing silt on adjacent areas. In silt samples from the first four floods, no TCDD was detected; however, TCDD just above the detection threshold (~1 ng/kg) was found in silt from the fifth flood.[20,24]

5.3. TCDD Levels in Atmospheric Particulates

TCDD transported by suspended air particulates (<100 μm) was monitored by high-volume samplers in June, 1977; however, on a regular basis, dustfall jars were extensively utilized to collect settling dust (>10 μm) (Fig. 4).[20,24,56] Monitoring was carried out to determine whether fine air dust from topsoil of the more contaminated areas was hazardous for the less contaminated or uncontaminated neighboring areas. As a preventive and risk management measure, atmospheric monitoring was implemented and tested in particular during the Zone A reclamation works of 1977–1980, since the latter involved mostly mechanical removal of topsoil layers (scarification). Of specific interest in this context, and for the amount of data available, are the results from settling air dust samples (July 1977–June 1980); the few findings from high-volume samplers—including measures of dust level (up to 0.14 mg/m^3)—showed general agreement with those of settling dust samples.

TCDD was detected sporadically and at different times by the dustfall jar network. Apart from Sampling Site (SS) 2 in the highly contaminated Subzone A_1, most individual findings were below the detection threshold (~0.1 ng/m^2 per day) and only seldom were detectable levels present; maximum values of 0.39 and 0.35 ng/m^2 per day were measured, respectively, at SS 1 in July, 1978, and at SS 6 in June, 1978. On the contrary, TCDD was detected in most samples from SS 2; peak deposition values of 0.75, 0.79, and 0.49 ng/m^2 per day were observed in September, 1977, July, 1978, and in the July 5–September 15 period of 1979, respectively. SS 2 showed seasonal variations in both dust and TCDD deposition rates, these normally reaching a peak during the summer.

In dust, TCDD levels were found to range between ~0.06 (detection threshold)[24] and 2.1 ng/g—the latter determined in the summer of 1977—with the higher values detected at SS 2 during the summer periods.[20,56] The TCDD amount in the dust decreased with increasing distance from areas with higher levels of contamination, so that airborne TCDD could not really significantly affect TCDD levels in the top layers of soil. The data from the dustfall jar at SS 6 did not fit into the above picture very well, probably because of a nearby municipal solid waste incineration plant.

In order to increase the sensitivity of detection, the individual sediment extracts from a sampling site were pooled and reanalyzed. Six or seven extracts of sequential samplings were employed for each pool. In most cases, the time-averaged TCDD levels resulted in values being below or near detection threshold (~0.01 ng/m^2 per day), although the 1978 March-through-October pools from SSs 1 and 6 provided values approximately six times as high. The highest value of 0.23 ng/m^2 per day was measured at SS 2 during the same period. A slowly declining trend in TCDD fallout was observed after 1978.

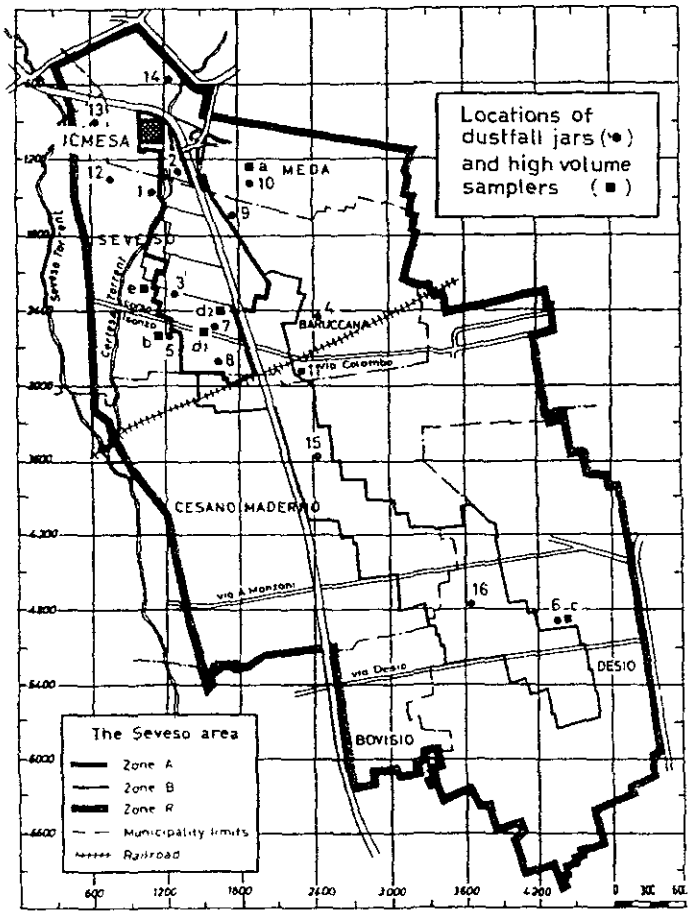

Figure 4. Network of dustfall jar and high-volume samplers utilized to monitor TCDD in air in the Seveso area. A dustfall jar is a passive collector of atmospheric precipitation and settling dust. Sampling periods normally lasted between 3 and 5 weeks. TCDD was assessed through the assay of sediment, whose amount was also determined after drying. No TCDD was ever detected in the aqueous matrix at a detection threshold of ~10 pg/liter. (Adapted from di Domenico *et al.*[56])

5.4. TCDD Levels in Plant Tissues

Shortly after the accident, samples of leaves, grass, and vegetables were collected in the area (especially in Zone A along the main diffusion pathway) and assayed. Many of these samples exhibited the effects of a severe chemical action, such as withering, burns, and yellow spots—effects that were less and less evident with increasing distance from ICMESA. As the south–southeasterly distance from the plant increased, TCDD levels in the vegetable samples decreased rapidly, following a negative exponential trend similar to that observed for topsoil (Fig. 3). For instance, in samples

collected in July, 1976, concentrations ranged between 50 and 0.01 mg/kg fresh tissue at distances of approximately 175 to 2000 m, respectively, from the source.[20]

Between 1978 and 1981, over 150 epigeal and hypogeal samples of several edible vegetable species (carrot, lettuce, onion, potato, and small radishes) from Zone R were assayed for contamination by TCDD and other TCDD isomers at a detection threshold of 0.2 ng/kg fresh vegetable tissue.[20] At levels close to 1 ng/kg, TCDD was identified only in two epigeal samples (relative frequency, 1.30%). In several cases (8.44%), the compound was detected at between 0.2 and 0.8 ng/kg, but this turned out to be unreliable because of uncertain identification. In nine cases (5.84%), analytical assessment could not be carried out because of interferences. In all of the other samples, TCDD was not detected. 1,3,6,8- and 1,3,7,9-TCDDs were quantitated in the 1.2–2.4 ng/kg range in epigeal samples (22.9%; 18.2% of the total set); however, they were not detected in hypogeal parts. Analysis of both types of samples provided negative outcomes in most cases (56.8%); uncertain results between 0.2 and 0.8 ng/kg accounted for the remaining determinations.

The levels of TCDD in wheat and oat grown in 12 experimental plots in Zone R were checked in 1980 by sampling ears with 10-cm stems during the maturation period.[20] Of 103 whole ear samples, not one contained detectable levels (>0.5 ng/kg) of TCDD, but 1,3,6,8- and 1,3,7,9-TCDD were detected with a frequency of 16.3% in the concentration ranges 0.76–2.2 and ~0.3–1.7 ng/kg, respectively. TCDD and its isomers were not found in 97 samples of wheat and rye kernels collected after harvesting.

In general terms, the above observations agree with a study by Wipf *et al.*[57] According to these authors, immediately after the accident, TCDD levels in samples of vegetable material from Zone A reached values up to some mg/kg; however, in the newly grown vegetation of the following years, TCDD levels were seen to drop by several orders of magnitude. In 1977, TCDD was not detected (<1 ng/kg) in the flesh of apples, pears, and peaches, nor in corncobs and kernels, all samples obtained from plants grown in a highly contaminated area; however, levels of the compound up to ~0.1 μg/kg were detected in the peel of the fruit and in the sheaths of the corncobs, this suggesting that contamination of vegetables was primarily by aerial route. Studies with carrots grown in a highly contaminated medium provided further evidence of the absence of a significant TCDD uptake from soil. No measurable amount of TCDD was detected in vegetable samples—including carrots—collected in Zone R in 1977–1980.

With respect to the Seveso accident, other studies performed on plants have been reported by Cocucci *et al.*,[58] Facchetti *et al.*,[59,60] and Sacchi *et al.*[61]

5.5. TCDD Levels in Wildlife and Domestic Animals

In the first few years after the accident, TCDD was detected in field mice and other wildlife specimens captured in areas characterized by considerable contamination (Table 6, upper section). In particular, chemical levels in the bodies of field mice were comparable to those in the soil, thereby providing some evidence for bioaccumulation. Earthworms were also tested as indicators of soil contamination. In 1980, samples of

Table 6
Impact of TCDD on Wildlife and Domestic Animals in the First Few Years after the Accident

TCDD levels in wildlife[a]

Animal	*N*	Tissue	Finding frequency	TCDD level (μg/kg) Mean	Range
Field mouse	14	Whole body	14/14	4.5	~0.07–49
Hare	5	Liver	3/5	7.7	2.7–13
Toad	1	Whole body	1/1	0.2	
Snake	1	Liver	1/1	2.7	
		Adipose tissue	1/1	16	
Earthworm[b]	2	Whole body	1/2	12	

Rabbit mortality[c]

Zone	Rabbits (*N*)	Deaths	Mortality (%)
A	1,089	348	31.9
B	4,814	426	8.8
R	18,982	1,288	6.8
Total	24,885	2,062	8.3

TCDD levels in rabbit liver[d]

Zone	Rabbits (*N*)	Finding frequency (%)	TCDD level (μg/kg) Mean ± S E[e]	Range
A	67	97	85 ± 12	3.7–630
B	19	84	90 ± 25	7.0–380
R	137	81	26 ± 6	0.27–460
Background[f]	86	13	13 ± 6	0.32–55

[a] From Fanelli *et al.*[62]
[b] Each sample was a 5-g pool of earthworms
[c] July 10–August 31, 1976. From Fanelli *et al.*[64]
[d] July 1976–July 1978. From Fanelli *et al.*[64]
[e] Standard error
[f] Surrounding areas

two earthworm species were collected in areas characterized by TCDD levels in soil of between ~0.06 and 9.2 μg/kg. It was observed that TCDD concentrations in the earthworm bodies were correlated with those in the soil; the earthworm bioaccumulation factor for TCDD from soil was estimated at ~10.[62,63]

In 1976, the entire Seveso area had been 81,000 domestic animals, many of which died within a short time following the accident. In Zones A, B, and R, animal breeding before the accident was mostly in family-size farms, where more than 80,000 poultry, rabbits, and other small animals were raised primarily for household consumption. At the time of the accident, there were also 349 cattle, 233 pigs, 49 horses, 49 goats, and 21 sheep. Cattle fed mostly on fodder made of grass, hay, and sliced corn collected

near the farms; during winter, the fodder was mixed with commercial feed. Pigs and poultry fed on commercial products from distant areas. Rabbits were given mainly grass harvested randomly, often from nearby farms but sometimes far away. After the accident, many rabbits died; to a lesser extent, mortality was observed in poultry and other small animals. These deaths began some days after the release of the cloud containing TCDD and the other chemicals; deaths increased remarkably in the following 2 weeks, and then decreased for months afterward.[63,64]

The total number of spontaneous deaths registered by the end of August, 1976, was 3281, of which 2062 were accounted for by rabbits (Table 6, middle section).[63,64] In particular, a mortality rate up to 100% was noted in animals fed on green fodder from contaminated areas, whereas a much lower mortality incidence was observed in animals on commercial feed or fodder collected before the accident or far away from ICMESA. During the first weeks, 2294 autopsies were carried out on animals (usually on rabbits or poultry) from nearly 1200 farms of the Seveso area. In addition to the normal incidence of known pathological signs, in ~15% of the farms on Zones A, B, and R many subjects showed pathological conditions never previously seen.

As the animal studies were being carried out, analytical detection of TCDD in the tissues of dead animals was also implemented to ascertain whether exposure to the chemical had occurred, establish a possible dose–response relationship, and determine if the rabbit could be used for biomonitoring owing to its capability to bioaccumulate TCDD in the liver (μg/kg range) before showing pathological signs (Table 6, lower section).[18,63,64] It should be mentioned that a comparative study on the uptake in the rabbit of TCDD in different formulations—including accident-contaminated Seveso soil—indicated that Seveso soilborne TCDD had a bioavailability on average 68% lower than solvent-borne (free) TCDD.[42]

Many of the farms kept dairy cattle, generally from five to ten cows. On average, daily milk production was ~10 liters/cow and destined for local consumption. Approximately 2 weeks after the accident, cows from Zone A were transferred to a special cowshed under sanitary surveillance. Cattle breeding in Zones B and R continued under veterinary control, but the animals were fed safe fodder from elsewhere. All of these cows were slaughtered in 1977 and 1978. In a first sampling campaign (July 27–August 28, 1976), milk was collected from cows living in areas at different distances from ICMESA, each farm usually providing a single sample obtained by pooling the individual milk samples from each cow of the farm.[63,65] The outcomes of this investigation proved that higher TCDD levels (from 76 up to 7900 ng/liter) were present in milk samples from farms close to the chemical plant; milk from outside Zone R and from the south end of Zone B did not contain detectable amounts of TCDD (<40 ng/liter) or contained TCDD at levels <200 ng/liter.[18,63,65]

5.6. Reassessment of TCDD Persistence Model in Soil

Several efforts have been made to investigate the TCDD temporal trend in Seveso soil. Early studies were based essentially on Zone A data obtained between the accident and 1979.[5,20,55,66,67] More recent studies have utilized data from Zone A as well

as from Zones B and R.[29,68] The subject has gained even more interest as the early TCDD figures for soil may have been affected by a large bias,[23,28,29] and yet they are critical for a correct interpretation of the chemical environmental dynamics. In particular, recovery yields were shown to never exceed 75% even when analytical procedures were at their best (since late 1979). On the other hand, Zone A findings before 1979 appeared to be affected by TCDD losses which, depending on the specific area and TCDD level in soil, might have reached values on the order of 70–90%.[23] However, accurate estimates of the analytical losses could not be provided because of the functional (but false) assumption used for model computing that no TCDD had disappeared from soil with time: this assumption has the effect of yielding optimistic loss figures.

In light of the above, the ongoing studies on the TCDD temporal trend in Seveso soil have the purposes of providing a more reliable description of TCDD persistence pattern, estimating analytical losses prior to late 1979, estimating the total amount of the compound released or, at least, deposited in the area hit, and possibly yielding new exposure and risk assessment figures for the people exposed. In fact, the assessment of TCDD environmental dynamics and levels in soil through time is critical to evaluating the long-term exposure of the once-exposed populations and the associated lifetime cancer risk.[35] Results obtained so far, relative to the first three points, are summarized below.

The data to serve as a basis for the current analysis were selected from the general data base according to stringent selection rules, and cover a time span of 5.5 years.[14,23] Figure 5 exhibits the TCDD time-trend regression function obtained by using a two-exponential model: analysis outcome details are summarized in Table 7.[14,69] According to the present model, the TCDD that remained after the period of rapid vanishing settled into the soil with little interaction with those environmental factors responsible for its initial disappearance. It is suggested that the initial (<0.5 year) rapid reduction

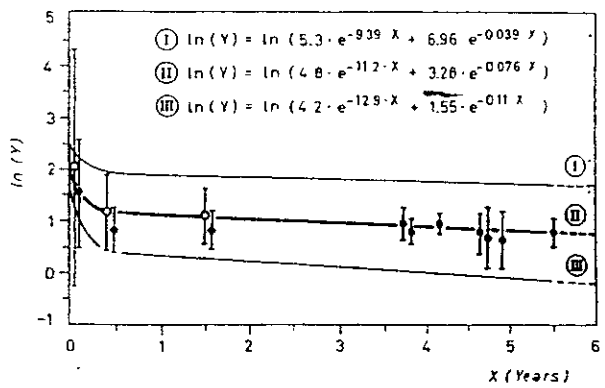

Figure 5. The two-exponential model developed to describe TCDD persistence in Seveso soil. The function has a steep downslope within < 0.5 year from the origin and then almost flattens out for longer times, in other words, in Seveso soil TCDD levels diminished rapidly shortly after blow-out, but did so at a much slower rate over the following 5 years. (Adapted from di Domenico *et al.*[69])

Table 7
Summary of Some Indicative Results of TCDD Persistence Studies in the Soil of the Seveso Area

Model function

$$\ln(Y) = \ln(q1 \cdot e^{-k3\,X} + q2 \cdot e^{-k4\,X}) \qquad q1 = Y^{o} \cdot k1 \qquad q2 = Y^{o} \cdot k2$$

Function parameters

Quantity	Mean estimate	Standard deviation	95% confidence limits	Units/comments
Fast dynamics[a]				
$q1$	4.8	2.5	4.2–5.3	Normalized[b]
$k3$	11.2	7.6	9.39–12.9	years^{-1}
$t^{1}/_{2}\{k3\}$	0.0619[c]		0.0537–0.0738[c]	years
Slow dynamics[b,d]				
$q2$	3.28	0.33	1.55–6.96	Normalized[b]
$k4$	0.076	0.022	0.039–0.11	years^{-1}
$t^{1}/_{2}\{k4\}$	9.1		6.2–17	years
Other estimates				
Y^{o}	8.0		5.7–12	Normalized[b]
$k1$	0.59		0.43–0.73	
$k2$	0.41		0.27–0.57	

Regression equations[e]

$\ln(Y) = \ln(5.3 \cdot e^{-9.39\,X} + 6.96 \cdot e^{-0.039\,X})$ Upper boundary
$\ln(Y) = \ln(4.8 \cdot e^{-11.2\,X} + 3.28 \cdot e^{-0.076\,X})$ Intermediate
$\ln(Y) = \ln(4.2 \cdot e^{-12.9\,X} + 1.55 \cdot e^{-0.11\,X})$ Lower boundary

[a] Arithmetic means and their standard deviations and confidence limits obtained from a specific simulation treatment [69]
[b] Reference to Cerlesi *et al.* [4]
[c] $\langle t^{1}/_{2}\{k3\}\rangle$ = 23 days, 95% confidence limits = 20–27 days
[d] Means and standard deviations from regression analysis
[e] Compare with curves of Fig. 5

was mainly related to the action of UV sunlight and heat-promoted volatilization on the TCDD which was then distributed very superficially on both vegetation and soil. Furthermore, the heavy rainfalls of the fall and winter of 1976 washed down the contaminated outer surfaces and disturbed the top layer of soil, moving TCDD deeper into soil and away from direct solar radiation, air streams, and precipitation. In the process, a dilution or loss of that fraction of TCDD loosely bound to the earth matrix occurred so that, after about half a year, only TCDD that was strongly bound to soil remained.

On these grounds, it has been concluded that two different half lives describe the TCDD persistence trend at Seveso: on average, they have been estimated to be 23 days (fast-vanishing dynamics) and 9.1 years (slow-vanishing dynamics). The equation representing the lower boundary of regression curve 95% confidence region provides an estimate of the total TCDD released (30–40 kg) which is fully compatible with what is known of the chemistry and physics of the accident. Based on such an equation,

these half lives assume the values of the corresponding 95% lower confidence limits (20 days and 6.2 years, respectively); further, the fast-vanishing dynamics appears to have been associated with the disappearance of most of the TCDD (~70%). Unfortunately, not much may be said as yet on how TCDD was released and distributed over the territory and, therefore, how biased the early contamination maps are.

Biodegradation was likely to be of negligible importance in TCDD reduction over the period explored (first 5.5 years).[70,71]

6. BIOLOGICAL DATA ON HUMAN EXPOSURE

Seven months after the ICMESA accident, a 55-year-old woman died of pancreatic adenocarcinoma, which had spread to the liver and to the extrahepatic bile ducts and had metastasized in the lungs. The subject had lived in an area of Zone A characterized by a mean TCDD level of ~200 $\mu g/m^2$. At the moment of the accident, the woman was eating inside her home with the windows and doors open; she also consumed vegetables from her garden in the 4 days following the event. The woman remained in her home until July 26. The first symptoms related to the tumor became clinically evident at the end of October 1976, and laboratory confirmation after exploratory laparotomy arrived a month later. As the subject had been exposed to the chemicals released from ICMESA by inhalation, ingestion, and dermal contact, TCDD presence in her tissues was investigated after death occurred. The following results were obtained and reported on a whole or wet weight basis, rather than the currently traditional lipid basis[72].

Fat	1840 pg/g
Pancreas	1040
Liver	150
Thyroid	85
Brain	60
Lung	60
Kidney	40
Blood	~6

These data provided a preliminary picture of TCDD distribution in human tissues following what could be defined as an "acute environmental exposure." Based on their findings, the authors suggested classifying tissues into four groups with, respectively, high (fat and pancreas), medium (liver), low (thyroid, brain, lung, and kidney), and very low (blood) levels of the chemical—the specific levels being eventually in association with preferential accumulation routes.

Approximately 10 years after the Seveso accident, analytical methods were improved and became available to measure TCDD levels in small blood samples.[73–76]

From the 1976–1985 laboratory medical examinations following the Seveso accident, there were some 30,000 1- to 3-ml serum samples which had been kept stored at −30°C since the time they were collected.[77–79] In 1988, a set of thirty 1976 serum and plasma samples was selected for TCDD assessment. The analytical results of this first set of determinations concern a selected group of ten Zone A children with chloracne, a